THE ETIOLOGY, PROGNOSIS, AND INDICATIONS FOR THE SURGICAL TREATMENT OF TUBERCULOUS PERITONITIS.[1]

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Etiology. *Frequency.* In 1825, Louis pronounced the dictum that chronic peritonitis is usually of a tuberculous nature. There is peritoneal involvement in 20 per cent. of tuberculous subjects (Wathen). In 4250 autopsies, Borschke[2] found 1393 cases of tuberculosis; of these 226, or 16 per cent., showed evidence of invasion of the peritoneum. Sixteen per cent. perhaps correctly represents the frequency with which tuberculous peritonitis occurs in tuberculous subjects in general at autopsy. Taylor Cummins,[3] reviewing 3405 autopsy records of the Pennsylvania, Philadelphia, and University Hospitals, found that 25.4 per cent. presented some type of tuberculosis. In addition, seventy-six records showed healed tuberculous foci. In 11 per cent. of the cases of tuberculosis there was peritoneal involvement. These are the "hard-worked statistics" referred to by Mayo,[4] and really convey very little additional information to us, since it is well known that somewhat over 60 per cent. of general autopsies reveal tuberculosis, active or healed. In 5687 intraperitoneal operations at St. Mary's Hospital, Rochester, Minnesota, 3 per cent. were for some variety of tuberculosis. From 1889 to 1900, Shattuck[5] found a record of 98 cases of tuberculous peritonitis treated in the hospital, in the medical and surgical sides.

Age. Tuberculous peritonitis is more frequently met clinically between the twentieth and the fortieth year; yet there are no exact limitations as to age. Fifty-six of ninety-eight patients studied by Shattuck were between the ages of fifteen and thirty. In children it is most common between the ages of two and four years. The common dicta regarding congenital tuberculosis are probably extreme. It is likely, however, that it is more common than is generally supposed. Miserocchi[6] found measles or pertussis, or both, in the antecedents of 83 per cent. of 352 tuberculous children.

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[1] Read at the meeting of the National Association for the Study and Prevention of Tuberculosis, Washington, D. C., May 17 and 18, 1906.

[2] Virchow's Archiv., 1892, Bd. cxxvii.

[3] Tuberculous Peritonitis; a Statistical Review, University of Pennsylvania Medical Bulletin, December, 1905, vol. xviii, No. 10, p. 272.

[4] Surgical Tuberculosis in the Abdominal Cavity, with Special Reference to Tuberculous Peritonitis, Journal of the American Medical Association, April 15, 1905, vol. xliv, No. 15, p. 1157.

[5] Prognosis and Treatment of Tubercular Peritonitis, The American Journal of the Medical Sciences, July, 1902, vol. cxxiv, p. 1.

[6] Abstract, Journal of the American Medical Association, April 30, 1904, vol. xlii, No. 18, p. 1115.

Rotch[1] gives some idea of its frequency in early life. He found that in eighteen years 69 cases were admitted to the Children's Hospital in Boston. Kissell[2] says that it is far more common in children than is supposed. In 883 collected autopsies upon tuberculous children, Biedert[3] found peritoneal involvement in 18.3 per cent., paralleling in frequency its occurrence in adults.

Sex. The Mayos state that at the operating table they find about four females to one male with tuberculous peritonitis. Osler[4] gives the ratio as 2 to 1. König[5] collected 131 cases, 120 in women, 11 in men. This is quite a reversal of the old teaching from autopsy findings, as from them it is somewhat more common in the male sex. I rather think that this disproportion in favor of females has been exaggerated. When the element of sexual activity has been eliminated, as in children, we find it about equally distributed in the sexes, as in the 69 children so carefully studied by Rotch, 39 boys and 30 girls.

For adults the correct proportion appears to be about as Osler states, two females to one male. Kelly[6] has shown that parturition is especially predisposing. On the other hand, hepatic cirrhosis, alcoholism, and tuberculous peritonitis are concomitant phenomena, more common in males, yet without any established sequence. Rotch and Mayo found a family history of tuberculosis in about 30 per cent. of their cases; other authors place it as high as 70 per cent.

The essential factor in the production of tuberculous peritonitis is Koch's bacillus; and as this parasite must be looked upon as a plant of relatively high development belonging to the actinomyces, of the group of oospora, it would, therefore, deserve the name of *Oospora kochii* (Roger[7]).

In the histogenetic study it is necessary to admit such elementary facts as the following: The transmission of tuberculosis from animals to man has been demonstrated sufficiently. The question whether tuberculosis may be transmitted from the lower animals to the human subject by means of meat, milk, etc., has lately been much discussed. Koch, the leader of the negative school, is opposed by Hamilton,[8] who has reinvestigated the subject, and he, with the majority of authors, now holds that tuberculous contagion is transmissible from man to man and from man to animals; from animals to man, and between animals of the same species.

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[1] Tubercular Peritonitis, Journal of the American Medical Association, January 10, 1903, vol. xl, No. 2, p. 69.

[2] Vratch, St. Petersburg, June 1, 1901.

[3] Jahrbuch f. Kinderh., 1884, xxi.

[4] Johns Hopkins Hospital Reports, 1890.

[5] Centralblatt f. Chir., 1884, No. 6, and Centralblatt f. Chir., 1890, No. 35.

[6] Operative Gynecology, 1898, vol. ii, p. 134.

[7] Infectious Diseases, 1903, p. 278.

[8] Quoted by Walsham, loc. cit.

Tuberculous peritonitis is one of the manifestations of lesions due to the action of Bacillus tuberculosis through one of its many channels of infection. The oft-repeated statement that tuberculous peritonitis is always secondary, is practically the unchallenged dictum emanating primarily from such authors as Veit, Nothnagel, Teleky, Vierordt, and a number of recent writers.

In view of conclusive proof furnished by Walsham,[1] in his prize essay, issued by the Royal College of Physicians of London, I believe that evidence will soon be forthcoming that the peritoneum may, like many other structures in the body, be the primary site of lodgement of Bacillus tuberculosis.

In seeking to trace the channel of infection of the peritoneum, we must bear in mind that the tubercle bacillus is immobile; that it is of slow growth, and that it produces very varied tissue changes in places where it lodges (Walsham). The channels of infection by the tubercle bacillus are classified as follows: (1) Hereditary transmission; (2) lymphatic vessels; (3) bloodvessels; (4) epithelial channels; (5) inoculation into the skin or tissues. We know that tubercle bacilli can pass intact mucous membrane and epithelium, and there are undoubted instances in which, with a normal placenta, tubercle bacilli may be found in the placental blood and in the fœtus. Chudovsky, in speaking of the etiology, says that there are cases known in which the fœtus becomes tuberculous. Martin[2] took particles from the lungs of a six and a half months' fœtus whose mother had died of tuberculosis and injected them into the peritoneal cavity of rabbits, with resulting death in four months. In another instance the blood from a five months' embryo whose mother had died of phthisis was inoculated into rabbits, with the result of tuberculous infection.

Schmoll[3] and Birch-Hirschfeld[4] found tubercle bacilli in the placenta and the capillaries of the liver in a child; the mother died of tuberculosis. Rindfleisch[5] found tubercles in the lungs of a child eight days old; the mother was tuberculous. The above cases show that tuberculous infection may occur during intra-uterine life through the circulation. The fœtus may be infected by extension of a tuberculous process in the tubes to the uterus and the fœtus contained therein. The spermatic fluid coming from a diseased testicle or epididymis may convey the infection.

Cnopp's[6] statistics show that out of 298 tuberculous children from a few days to twelve years of age no less than 147 had bone or joint tuberculosis. Only 8 of these showed evidence of visceral tuberculosis. Baumgarten[1] holds that the accidental conveyance of the tubercle bacilli to these parts would not account for such a large proportion of cases, and he expresses the view that the bacilli have been present since birth and have developed when the conditions became favorable. Mafucci[2] has shown that after infecting eggs with avian tuberculosis, the disease may remain latent in the chick for weeks or months; this is in support of Baumgarten's view, and these facts contain practically all that we can say upon the subject of inherited tuberculosis.

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[1] Channels of Infection in Tuberculosis.

[2] Quoted by Sir Dyce Duckworth, Tuberculous Peritonitis, The Clin. Jour., London, 1903, vol. xxii, 209.

[3] Quoted by M. Chudovszky, Tuberculous Peritonitis, Pester Med.-Chir. Presse, 1901, vol. xxxvii, No. 54.

[4] Ibid.

[5] Ibid.

[6] Quoted by Walsham, loc. cit.

The process of eruption of metastatic tubercles is called hematogenetic miliary tuberculosis. The blood channels have always been accredited with being a common mode of disseminating tuberculous infection. The bacilli may enter the blood current by way of the lymphatics, the thoracic duct, and the venous system, or they may gain entrance to the circulation through direct rupture of a bloodvessel. The small bloodvessels about an infected focus are always involved and become thrombosed; hence, they are not the carriers of the infection. As Weigert[3] and Benda[4] have shown, a general miliary eruption of tuberculosis is consequent upon tuberculous disease of the walls of the large bloodvessels. Under these circumstances, miliary tuberculosis may develop in the peritoneum, as in other organs or serous membranes.

The next path of infection that we shall consider is the lymphatic. Sydney Martin has shown that the bacilli can pass through healthy intestine to the mesenteric glands without leaving visible evidences upon the surface; or through open ulcers—an infrequent but well-recognized mode of infection.

Professor Benda, at the meeting of the British Congress upon Tuberculosis, spoke thus: "Whether tubercle bacilli can gain access through intact mucous membrane is a matter extremely difficult to determine by methods of pathological anatomy; we find a large number of bronchial glands tuberculous, in which we do not discover a trace of any bronchial lesion." Walsham says: "From some observations of my own upon tonsillar and intestinal tubercle, I believe that the tubercle bacillus can be passed through a normal intact membrane without showing the atrium of infection." Or, as Professor Hamilton expresses it, "Can the absorbents of mucous membrane seize upon the bacillus and hand it on to the corresponding lymphatics without becoming tuberculous?" He answers his own interrogation as follows: "My own impression is that they can; that the active point of invasion may, from local conditions, prove unfavorable to the development of tubercle and the bacillus is passed through them to a tissue more favorable for its growth." With this conclusion, Walsham agrees, after repeated observations upon tuberculous bronchial and cervical glands. He holds that it is certain that they receive their infection through the normal, intact epithelium of the trachea, bronchii, or œsophagus. The older theory that the bacilli pass through the lung and thus reach the bronchial glands is improbable.

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[1] Quoted by Walsham, loc. cit., p. 29.

[2] Ibid., p. 30.

[3] Ibid.

[4] Ibid., p. 37.

It has been demonstrated by Dr. Litterer, of Nashville, Tennessee, that mesenteric glands may become infected through the intestines (proved microscopically and by guinea-pig inoculation) without lesion to the wall of the gut. A very interesting question, which he has been seeking to elucidate, is the possibility of primary infection of the peritoneum from the alimentary canal without involvement of the bowel wall, or of the mesenteric or retroperitoneal glands. In other words, can we have in this way a primary infection of the peritoneum? This has not been demonstrated; but there are many cases of tuberculous peritonitis in which it is impossible to discover a primary focus.

Reasoning by analogy from Walsham's investigation in infection of the bronchial and cervical glands, may not the infection reach the peritoneum through the normal bowel wall? It must be admitted that tuberculous peritonitis usually arises secondarily to tuberculosis of the pleura, abdominal or pelvic organs, so that the continuous extension of the process is clearly demonstrable. Sometimes, however, this relation cannot be traced; in which event, no doubt, the infection has been carried from some distant focus by the flood or lymphatic vessels.

In 226 cases of peritoneal tuberculosis, Borschke failed to find a primary focus in only 2 cases. The constancy of a primary source of infection in tuberculous peritonitis is noted by Primbram,[1] who states that out of 165 autopsies, 87 cases were attributable to intestinal tuberculosis, 65 to primary and glandular disease, 8 to tubal and uterine, and 5 to osseous tuberculosis. Taylor Cummins, who has studied this relation so closely, states that in his series of cases, 84 per cent. were complicated by pulmonary disease, and 32.6 per cent. by intestinal disease.

The female generative organs, especially the Fallopian tubes, may be the starting point of the infection to the peritoneum. From there the infection may be by direct inoculation or through the medium of the lymph channels. It has been said that postmortems upon females showed from 25 to 50 per cent. of tuberculous cases with tubal disease. Murphy[2] and Mayo believe that the common source of infection in females is the genital tract, especially the tubes. Osler considers tuberculous peritonitis in women a common sequence of tuberculous salpingitis. Upon the other hand, Williams[1] asserts that tuberculosis of the Fallopian tubes is far more frequently the result than the cause of peritoneal tuberculosis. That infection from without, through the genitals, is a mode of invasion seems established.

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[1] Quoted by Taylor Cummins, Tuberculous Peritonitis; A Statistical Review, University of Pennsylvania Medical Bulletin, December, 1905, vol. xviii, No. 10, p. 272.

[2] Tuberculosis of the Female Genitalia and Peritoneum, American Journal of Obstetrics, 1904, vol. xlix, No. 2, p. 6.

I have it upon the authority of Berkeley[2] that Morgagni, in 1744, was the first to record a case of genital tuberculosis. Literature is plethoric with the report of cases from Louis[3] in 1825, to Pinner[4] and Weigert in 1880, all taking the view that the infection is ascending; that is, from the genitals to the peritoneum. In 1880, Pinner found that powdered cinnabar placed in the peritoneum of rabbits could be found in the vagina in a very short time, being conducted from the peritoneum by the current of the tubal cilia; and it is presumed that in the same way tubercle bacilli can be wafted out of the peritoneal cavity into the genital tract. Of course, it is understood that they got into the peritoneal cavity by their natural carriers, the leukocytes, from the bowel or through the medium of the lymphatic channels from some primary focus, and it is remarked that, favoring this method of infection, the pelvis being the lowest portion, tubercle bacilli gravitate to that region.

The causal relation existing between peritoneal tuberculosis and tuberculosis of the genital organs is somewhat misty. For a long time it was believed, in obedience to Pinner and Weigert's theory of gravitation, that the peritoneum was the source of genital tuberculosis. Murphy, in contending for this theory of gravity, comments upon his experiments which appear to confirm it, as follows: "Tuberculous products placed in the peritoneum of the monkey settle in the pelvis and are not carried by the lymphatic current in the direction of the diaphragm." These conclusions run counter to Muscatello[5] and Clark's investigations of peritoneal currents.

Against any theories we may entertain as to whether this infection is ascending or descending, is the clean-cut statement of facts by Mayo: "Having under observation a small number of patients in whom simple laparotomy had failed in the permanent cure of tuberculous peritonitis, we began to do a radical operation, performing hysterectomy with removal of the ovaries and tubes. The condition of the uterus and ovaries on examination did not justify so mutilating a procedure, the tubes showing unmistakable evidence that they alone were the source of infection. It became clear that in tuberculosis of the peritoneum, in a very large majority of women,

[1] Johns Hopkins Hospital Reports, 1892, vol. iii.

[2] Genital Tuberculosis in the Female, Journal of Obstetrics and Gynecology of the British Empire, January, 1903, vol. iii, p. 31.

[3] Quoted by Berkeley, Genital Tuberculosis in the Female, Journal of Obstetrics and Gynecology of the British Empire, January, 1903, vol. iii, p. 31.

[4] Quoted by Berkeley, Ibid.

[5] Virchow's Arch., 1895, vol. cxliii.

a lupus of the mucous membrane of the tube was a source of infection to the peritoneum."

Mayo, dissenting from the idea of gravity advanced by Pinner and adopted by many, says: "The peritoneal involvement is greatest in extent near the seat of local infection. This has been generally noted, and heretofore ascribed to gravity. It is more likely due to the proximity to the seat of infection." I regret my inability to express myself definitely upon this very essential point. The question is raised, but unanswered by all of the three great classical writers upon genital tuberculosis, Berkeley, Williams, and Murphy.

Understanding the nature of the tubercle bacilli, it is quite reasonable to assume that the Fallopian tubes may become tuberculous by ascending infection from the lower genital or urinary tract; or from drifting bacilli in the peritoneum. The causal agent of tuberculous peritonitis, the tubercle bacilli, may infect the peritoneum from the tubes, through the medium of the lymphatics or by direct inoculation. Tuberculous peritonitis occurs from the male genital organs along the lymphatic vessels of the spermatic plexus or through the ampullar end of the vas deferens.

PROGNOSIS. Prior to König's first report in 1884, tuberculous peritonitis was regarded as an almost uniformly fatal disease when treated medically. The effects of the operative treatment have been to teach us that peritoneal tuberculosis is often present without producing special symptoms, and with a natural tendency to recover more often than is even now conceded. While the prognosis is not altogether bad, the gravity of an acute peritoneal infection, with or without recognizable primary lesion, cannot be overestimated. If we hold that peritonitis of this special variety is secondary to visceral or glandular infection, the peritoneal involvement may be arrested or controlled; but the ultimate prognosis will rest upon the activity of the primary lesion.

Peritoneal tuberculosis, appearing as a part of a general miliary tuberculosis, offers but little hope for even a temporary arrest. An acute peritoneal involvement, though localized, may become rapidly general and be attended with such toxic symptoms that we should regard it with the greatest apprehension.

While König taught us the value of the operative treatment he was among the first to advocate a selection of cases, and to condemn laparotomy as a uniform procedure in all types of peritoneal tuberculosis. Medically treated this affection is amenable to cure. Schröder[1] in his medical clinic obtained about 41 per cent. of cures. Rose,[2] who cites articles by Borchgrevink,[3] who strongly advocates expectant treatment over surgical measures, gives these rather startling statistics: of 44 cases of tuberculous peritonitis 22 were treated by operation, with 64 per cent. of recoveries; 22 without operation, with 82 per cent. of recoveries, 68 per cent. of which were of over a year's standing. Spath[1] long ago advocated conservatism, but not to the exclusion of surgical treatment. Caille,[2] in a strong article, shows the futility of medical treatment of tuberculous peritonitis in children.

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[1] Quoted by Walsham, Channels of Infection in Tuberculosis.

[2] Mittheil. aus den Grenzgeb. der Med. u. Chir., 1901, Bd. viii.

[3] Fall von anatomisch nachgewiesenes Spontanheilung der tuberkulösen Peritonitis, Deutsch. med. Woch., 1903, xxix, 43–45.

In uncomplicated subacute or chronic cases the outlook is distinctly favorable. In very young children with a strong inherited predisposition and severe infection the outlook is distinctly bad. The symptoms that affect the prognosis are: continuous fever, rapid wasting, persistent diarrhœa, and recurring exacerbations. The complications that make prognosis unfavorable are: pleurisy, localized suppurations, caseation of the mesenteric glands, and intestinal obstruction. Multiple sites of localization forecast a fatal ending.

The subacute and chronic ascitic forms are the most amenable to operative treatment. In only exceptional instances of complications and for the relief of special conditions should operation be undertaken in the fibrous form. The ulcerous form offers an unfavorable outlook, yet even these cases may eventuate in cure. After five different operations upon one patient, extending through a period of two years, I had the satisfaction of seeing absolute cure follow from drainage of many caseous masses; the patient is now a grown woman, in perfect health, and the mother of children.

All forms except the peracute are at least temporarily benefited, and a very conservative estimate of absolute cures can be placed at about 50 per cent. It will be a very difficult matter to eradicate from the active surgeon's mind these established benefits of the operative treatment of tuberculous peritonitis, and the day has not yet arrived when "surgery must hand back serous tuberculous peritonitis to the internal medicine clinic with thanks for the splendid opportunity which a misunderstanding gave to the profession by means of laparotomy, to study tuberculosis in one of the large cavities of the body." (Borchgrevink, Fenger.)

CONTRAINDICATIONS TO OPERATION. In general miliary tuberculosis, medicinal hygienic measures should be employed. Dry, fibrous peritonitis presents the greatest difficulties in operation, and as it indicates a stage in the process of healing, surgical measures should not be employed except to meet complications, such as intestinal obstruction. In the ulcerous form, with great emaciation and prostration, operation is contraindicated. Lindfors holds that the co-existence of pleurisy darkens the prognosis. Active lesions in other parts are contraindications to operation. Intervention is specially indicated when it appears that the peritoneal tuberculosis is the only active manifestation. In peracute cases, those of violent onset that resemble appendicitis or intestinal obstruction, the expectant treatment should be preferred to operation.

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[1] Deutsch. med. Woch., 1889, No. xx.

[2] Archiv. Ped., July, 1900.

SURGICAL INDICATIONS. Fenger's[1] pronounced teaching against surgical interference in the ascitic form is met by the equally radical statement of Dennis[2] that tuberculous peritonitis has been taken out of the realm of internal medicine and transferred to clinical surgery. I must dissent from both these extreme views. If one author, Herringham,[3] observed in the course of five years 25 cases diagnosed as tuberculous peritonitis, of which 19 recovered without operation and only 3 were unimproved; and if so careful a clinician as Shattuck is lead to conclude from an analytical study of 98 cases that tuberculous peritonitis under purely medical treatment may be followed by apparently complete recovery, even if complicated by tuberculosis elsewhere, we should surely hesitate before declaring this to be exclusively a surgical condition.

It is exceedingly fallacious to assume that the recognition of tuberculous peritonitis or a supposed diagnosis of that condition calls for immediate operation. In the exudative form we know that absorption takes place in those cases in which there is marked inprovement in the condition of the patient; resorption of the exudate can only take place when the functions of the body are in vigorous activity. It is obvious, then, that should medical and hygienic measures fail to establish this standard of good health operative interference is demanded.

Too frequently it is stated that simple incision is sufficient. The exudate must be completely removed. In the early stage this is especially necessary, for if the peritoneum is not immune the retained bacilli and their toxin keep up the peritoneal inflammation and the ascites will, therefore, reform. So in chronic cases, if some of the fluid is left, although the peritoneum may be somewhat immune, the irritation caused by retained fluid will lead to a re-accumulation of the effusion. Obviously, then, laparotomy is necessary for complete removal of the exudate. All the fluid connot be removed by mere puncture. Drainage is not desirable; indeed, it leads to infection and both tube and gauze drainage may produce fecal fistula. Neither is it shown that irrigation is essential. Efforts should not be made to detach adherent viscera further than to free encapsulated fluid or to relieve obstruction. Germicidal irrigations and local applications are of very doubtful value. Enlarged caseous mesenteric glands may be enucleated, though this is often a difficult task.

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[1] Treatment of Tuberculosis of the Peritoneum, Annals of Surgery, 1901, vol. xxiv, p. 771.

[2] The History and Development of Surgery during the Past Century, American Medicine, February 11, 1905, vol. ix, No. 6, p. 227.

[3] Quoted by Joseph Eichberg, Some Clinical Aspects of Tubercular Peritonitis, Journal of the American Medical Association, 1903, vol. xli, No. 41, p. 829.

Guided by the teaching of Murphy and Mayo, we all recognize the importance of removing foci of infection when possible. This should invariably be done when the appendix or Fallopian tubes is the source of supply.

If the serous covering of these organs shows miliary tuberculosis merely as a part of general peritoneal tuberculosis it is useless to remove them. It is well to follow the general law established by Ochsner, "Remove the tuberculous tissue if the section can be made through healthy parts." We should exercise the greatest care in all our intra-abdominal manipulations; avoid abrasion and injury to the serous covering of the viscera. The general conclusion is justified that tuberculous peritonitis can be cured by laparotomy. The unsuccessful cases are those that have advanced disease in other organs. The probable rationale of cure is that the manipulation and exposure cause an afflux of normal serum which is antitoxic. We should not hesitate to subject the patient to repeated operations if there is re-accumulation of fluid, especially if the first operation has been early in the progress of the disease.

OPERATIVE PROGNOSIS. Operative results give a recovery rate of from 25 to 30 per cent. The immediate operative mortality is but slightly over 3 per cent.; Mayo lost 3 cases out of 89. Cheyne says that in 385 operations 53 per cent. of the patients were well two years after operation. Rorsch[1] presents statistics of 358 patients treated operatively, 250 (70 per cent.) of whom were cured; 118 were under observation six months, 79 one year, and 53 two years.

Mayo contends strongly that tuberculous peritonitis has its origin in a local focus, and that the ultimate prognosis depends upon our ability to remove the primary site of disease. In 26 cases due to tubal infection in which he did the radical operation, 25 recovered. Several of these had had previous operation with recurrence. The prognosis after treatment by both medical and surgical methods, has been carefully studied by Shattuck. Out of the 98 cases treated the end-results are known in 57 cases; 25 of these were treated medically, the ultimate mortality which reached to a period of eleven years is 68 per cent.; 32 cases surgically treated, mortality 37.5 per cent. From these figures we learn that tuberculous peritonitis may recover under simple medical treatment; and evidence is not wanting to show that an anatomical restoration may be obtained.

Tuberculous infection of the parietal wound does not occur. Pyogenic infection is infrequent. A complication to be greatly feared is the formation of a fecal fistula; it will not occur in the ascitic form unless drainage is employed; it is especially likely to follow in the ulcerous form. The fistulæ are very obstinate; in Czerney's clinic in 3 out of 4 cases he failed to close the fecal fistula by suture. I have had the same mortifying experience in 2 cases.

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[1] Revue de Chirurg., 1893, vol. xiii.